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5.0 RISK CHARACTERIZATION FOR NON-LEAD CHEMICALS

Risk characterization is the summarizing step of risk assessment (USEPA 1995d). In the risk characterization, the toxicity values (RfDs and SFs) are applied in conjunction with the concentrations of COPCs and intake assumptions to estimate cancer risks and health hazards other than cancer.

Noncancer health hazards and cancer risk were calculated for both the reasonable maximum exposure (RME) and central tendency (CT) exposure conditions. RME hazard/risk estimates are based on the maximum exposure that is reasonably expected to occur at a site. Intake parameter values were selected so that the combination of all parameters resulted in an estimate of the RME for a particular exposure pathway. By design, the estimated RME is higher than that expected to be experienced by most of the exposed population. As recommended in EPA's *Guidance for Risk Characterization* (USEPA 1995d), CT exposure estimates reflect the central estimates of exposure or dose. The CT exposure estimate is intended to be more representative of average exposures.

5.1 METHODOLOGY FOR ASSESSING NONCANCER HAZARD

The potential for adverse health effects other than cancer (noncancer effects) was characterized by dividing estimated chemical intakes (Appendix A, Table 7 through 10 series) by chemical-specific RfDs (Appendix A, Table 5 series). The resulting ratio is the hazard quotient (HQ), derived as follows:

Use of the RfD assumes that there is a level of intake (the RfD) below which it is unlikely that even sensitive individuals (e.g., senior citizens and children), will experience adverse health effects over a lifetime of exposure. EPA Region 10 recommends that, if available, the child-specific RfD be used when calculating child hazards. Otherwise, use of the chronic RfD along with the average child chemical intake is appropriate (USEPA 1999g). If the average daily intake exceeds the RfD (that is, if the HQ exceeds 1), there may be cause for concern regarding noncancer effects (USEPA 1989).

The EPA risk assessment guidelines (USEPA 1989) consider the additive effects associated with simultaneous exposure to several chemicals by first specifying that all hazard quotients be summed across exposure pathways and chemicals to estimate the total hazard index. This summation conservatively assumes that the toxic effects of all chemicals would be additive, or in other words, that all chemicals cause the same toxic effect and act by the same mechanism (USEPA 1986).

If the total hazard index is less than or equal to 1, multiple-pathway exposures to COPCs at the site are considered unlikely to result in an adverse effect. If the total hazard index is greater than 1, further evaluation of exposure assumptions and toxicity, including consideration of the specific

affected target organs and the mechanisms of toxic actions of COPCs, is warranted to ascertain whether the cumulative exposure would in fact be likely to harm exposed individuals.

5.2 METHODOLOGY FOR ASSESSING CANCER RISK

The potential for carcinogenic effects is evaluated by estimating the probability of developing cancer over a lifetime based on exposure assumptions and chemical specific toxicity criteria. The increased likelihood of cancer due to exposure to a particular chemical is defined as the excess cancer risk (i.e., in excess of a background cancer risk of 1 in 100 or 1 x 10⁻²). Excess lifetime cancer risk is estimated by multiplying the estimated chemical intake by the cancer SF, as follows:

Cancer Risk = Chemical Intake (mg/kg-day) x SF (mg/kg-day)⁻¹

This formula applies to cancer risks lower than 1×10^{-2} (1 in 100). All cancer risks in this assessment were lower than 1×10^{-2} .

The risks resulting from exposure to multiple carcinogens are assumed to be additive. The total cancer risk is estimated by adding together the estimated risk for each COPC and for each exposure pathway (Appendix A, Table 8 through 10 series). The EPA's target acceptable excess cancer risk range is 10^{-6} to 10^{-4} (USEPA 1991c). Cancer risks which fall within this range will be further evaluated in the FS where risk management decisions will be considered.

5.3 RISK CHARACTERIZATION RESULTS

The results of the risk calculations are provided in Appendix A, Table 7 through 10 series. Table 5-1 provides CT and RME values for total hazard index and cancer risk for each exposure scenario. In addition, the table provides information regarding key chemicals and media contributing to RME hazard index and cancer risk values. Figures 5-1 to 5-20 also provide information regarding key chemicals and media contributing to the RME hazard index and cancer risk. The following subsections discuss noncancer hazards, cancer risks, and hazard/risks for scenario combinations.

5.3.1 Noncancer Hazard

This section discusses total hazard indices for chemicals and pathways in each exposure area, and chemical-specific hazard indices for risk drivers summed across pathways in each exposure area. Noncancer hazards were evaluated for four age groups: child residential and public recreational (child visitor), 0 to 6 years; child neighborhood recreational, age 4 to 11; child/adults, age 0 to 30; and occupational adults (construction workers), 25 years of exposure. In all cases, the greatest hazards were for children, age 0 to 6.

Total Hazard Indices

As shown in Table 5-1, total CT hazard indices were less than or equal to 1, with the exceptions of the child residential scenario in the Side Gulches (CT hazard index = 2) and the future residential scenario in Burke/Nine Mile (CT hazard index = 10 and 5 for child and child/adult,

respectively). Total RME hazard indices exceeded 1 for the 0- to 6-year age group in all of the geographical areas where residential hazards were evaluated: the Lower Basin, Kingston, the Side Gulches, Osburn, Silverton, Wallace, Mullan, and Burke/Nine Mile. Total RME hazard indices also exceeded 1 for a number of other scenarios: the future residential scenario in Burke/Nine Mile; the child public recreational scenario in the Lower Basin and Kingston; the child/adult residential scenario in the side gulches; the child neighborhood recreational scenario in the Side Gulches and Burke/Nine Mile; the occupational scenario in the Lower Basin (Figure 5-1);the future child/adult residential scenario in Burke/Nine Mile; and the vegetable garden pathway.

- ! For residential scenarios, the key medium contributing to total RME hazard indices was yard soil. One exception was tap water in the Side Gulches, which also contributed significantly to the total RME hazard indices (Figures 5-2 and 5-3).
- ! For residential scenarios, arsenic and iron were the key chemicals contributing to total RME hazard indices (Figures 5-4 and 5-5).
- ! In the special case of future use of groundwater by residents in the Burke/Nine Mile area, cadmium and zinc were the key chemicals contributing to the total RME hazard indices (Figure 5-6).
- ! Depending on the exposure area, one or more of various media (upland surface soil, soil/sediments, sediments, and waste piles) were key contributors to total RME hazard index for recreational scenarios (Figures 5-7 and 5-9).
- ! For recreational scenarios, arsenic and iron were the key chemicals contributing to total RME hazard index (Figures 5-8 and 5-10).
- ! For occupational scenarios, arsenic and iron were the key chemicals contributing to total RME hazard index (Figure 5-11).
- ! For homegrown vegetables, cadmium was the key chemical contributing to the total RME hazard index, whereas for fish, mercury was the key chemical (Figures 5-12a-b).

The total RME hazard indices suggest that several of the exposure scenarios listed could pose a threat of noncancer health effects, assuming that the effects from the key COPCs (i.e., arsenic and iron or cadmium and zinc) are additive. As discussed in the following section, this is a protective assumption that probably overestimates the hazard index at the site.

Chemical-Specific Hazard Indices

Risk drivers in each of the current scenarios were either (1) arsenic and/or iron or (2) cadmium and/or zinc. There is no evidence that noncancer effects from arsenic plus iron or cadmium plus zinc are additive. On the contrary, it is more likely that the interactions of these two pairs of chemicals are antagonistic (protective), rather than additive. Iron is known to interfere with the

absorption of ingested metals (e.g., calcium, lead, and cadmium); (whether iron affects the absorption of ingested arsenic is unknown). Oral zinc supplementation decreases the absorption of orally administered cadmium in humans (ATSDR 1999a). Dietary zinc reduces cadmium-induced testicular damage, hypertension, pulmonary damage, and developmental effects in animals (USEPA 1994c). The mechanism of zinc's reduction of cadmium toxicity may be (1) a zinc-induced decrease in the bioavailability of ingested cadmium and/or (2) a decrease in cadmium/zinc ratio in target tissues (USEPA 1994c). Chemical interactions are further discussed in Section 7.

Because of the uncertainties surrounding chemical interactions, chemical specific hazard indices were also evaluated and are discussed below. Table 5-2 lists scenarios for which hazard indices for specific chemicals (added across pathways) exceeded 1.

For chemical-specific RME hazard indices:

- ! Other than the special cases (discussed in the last two bullets below), the only specific chemicals with RME hazard indices that exceeded 1 were arsenic and, in one case only (residential exposures for children 0 to 6 years in the Lower Basin), iron.
- ! Of the nine exposure areas, four (Kingston, Silverton, Wallace, and Blackwell Island) had no cases in which specific chemicals had RME hazard indices that exceeded 1.
- ! Only residential scenarios had RME hazard indices that exceeded 1. In no cases did the neighborhood recreational, public recreational, or occupational scenarios have chemicals with RME hazard indices that exceeded 1.
- ! Tap water in the Side Gulches was the only medium other than soil for which an HQ exceeded 1, with the exception of the special cases described in the last two bullets.
- ! In the special case of future use of groundwater in the Burke/Nine Mile area, RME hazard indices for cadmium and zinc each exceeded 1 for the residential child and residential child/adult scenarios.
- In the special case of homegrown produce, RME hazard indices for cadmium exceeded 1 for the residential child/adult scenarios (vegetable hazards were not calculated separately for the child age group because ingestion rates for this parameter were age adjusted and apply equally to both age groups).

The hazard index for iron exceeded 1 only in the Lower Basin, and the Lower Basin is the only area where iron concentrations are likely to be greater than background concentrations. Therefore, iron is a concern only for the 0- to 6-year age group in the Lower Basin, while arsenic concentrations are a concern in most areas. Arsenic is the significant noncancer chemical besides lead in the entire Basin.

Hazards in the Side Gulches from the tap water are due to arsenic concentrations of 8 μ g/L in one private well. In general, arsenic concentrations in private sources from all areas are the major contributors to drinking water hazards. Most private water sources had concentrations ranging from 1 to 4 μ g/L, with the Side Gulches well containing the highest concentration in the Basin. The MCL for arsenic is expected to be lowered from 50 μ g/L to either 3, 5, or 10 μ g/L. Depending on the new limit, arsenic concentrations in some private water sources in the Basin may exceed the new MCL for arsenic. In general, arsenic concentrations in public water systems ranged from nondetected to 0.6 μ g/L.

The results of the noncancer evaluation for RME cases suggest that the following exposure scenarios could pose an unacceptable threat of noncancer effects if current conditions remain the same:

- ! Arsenic and iron (primarily in yard soil) for child residents in the Lower Basin,
- ! Arsenic (primarily in yard soil and tap water) for child and child/adult residents in the Side Gulches (note: arsenic concentrations from many private sources throughout the Basin may exceed the anticipated new MCL),
- ! Arsenic (primarily in yard soil) for child residents in Osburn and Mullan, and arsenic in soil and groundwater in Burke/Nine Mile (this groundwater is not currently used as a drinking water source),
- ! Cadmium and zinc in groundwater for child and child/adult residents in Burke/Nine Mile, and
- ! Cadmium in homegrown vegetables for child/adult residents.

For the CT cases, potential unacceptable exposures occur only for child residents in the Side Gulches when all exposure routes are combined and for future child and future child/adult residents of Burke/Nine Mile from ingestion of tap water.

5.3.2 Cancer Risks

Cancer risks were evaluated for two age groups: child/adult, age 0 to 30, and occupational adult, 25 years of exposure.

As shown in Table 5-1 and Figure 5-13, total RME cancer risk for each scenario was in the range of 10⁻⁶ to 10⁻⁴. CT cancer risk for each scenario was also in or below the range of 10⁻⁶ to 10⁻⁴ (Table 5-1 and Figures 5-16 through 5-18). The risk values presented in the tables and the text are rounded to one significant figure as recommended by the EPA (USEPA 1989). The unrounded values are shown in the figures and in Appendix A, Table 8 through 10 series.

Arsenic was the only carcinogenic chemical evaluated at the site. For the residential scenarios, exposure to arsenic in yard surface soil contributed most of the total RME cancer risk (Figure 5-14). Arsenic in tap water also contributed significantly to total RME cancer risk for

residents at the Side Gulches (see Figure 5-14 and discussion in previous section). Although tap water was not the primary contributor to cancer risk for the residential scenarios, RME cancer risk for tap water exceeded 1×10^{-6} in all exposure areas (Figure 5-14).

For the special case future residential scenario at Burke/Nine Mile, groundwater contributed approximately 20 percent of the total RME cancer risk (Figure 5-15). Arsenic risks in surface/subsurface soil for construction workers ranged from 3 x 10⁻⁵ to 1 x 10⁻⁴ (Figure 5-18). For recreational scenarios in each exposure area, the following media contributed to most or all of RME cancer risk due to arsenic (Figures 5-16 through 5-18):

- ! Soil/sediment in the lower Coeur d'Alene River for the Lower Basin (highest concentrations of arsenic in the entire Basin with the exception of waste piles),
- ! Soil/sediment at the NS confluence in Kingston,
- ! Upland surface soil from the Elk Creek area and sediment from Elk Creek Pond in the Side Gulches (Elk Creek area soil and sediment had the second highest arsenic concentrations in the entire Basin after floodplain soil/sediments in the Lower Basin),
- ! Sediment in the South Fork (Osburn, Wallace, and Silverton neighborhood exposures),
- ! Surface soil from waste piles in Burke/Nine Mile,
- ! Soil in waste piles and sediment in the South Fork in Mullan, and
- ! Soil/sediment from the Spokane River on Blackwell Island.

Although surface water was never the primary contributor to RME cancer risk, cancer risk estimates exceeded 1 x 10⁻⁶ for surface water in the Lower Basin (neighborhood and public recreational), Kingston (public recreational NS confluence exposures), and the Side Gulches (neighborhood recreational Elk Creek Pond exposures) (Figures 5-16 and 5-17). The samples from these areas consisted of "disturbed" surface water (see Section 2.8), that is, surface water that contained suspended sediments due to disturbance by the sampler. Therefore, surface water risks in the other water bodies could have been as high as those seen for the "disturbed" water bodies if the sampling methods had been the same for all water bodies.

5.3.3 Hazards/Cancer Risks for Combinations of Scenarios

The hazards/cancer risks for individual scenarios discussed in the previous sections do not consider the potential for the same individual to be exposed via more than one exposure scenario. For example, it is possible that children and adults exposed to yard soil and tap water at their home could also be exposed to other media (soil, sediment, waste piles, or surface water) during recreational use of schools, parks, creeks, and ponds in the neighborhood. Other combinations of scenarios are also possible such as residents who also visit public recreational areas, catch and

eat locally caught fish or residents who also eat homegrown vegetables, and so on.

Child/Adult Residential Plus Neighborhood Recreational

Table 5-3 and Figures 5-19 and 5-20 provide total RME hazard index and cancer risk for the combined residential child/adult and neighborhood recreational scenarios. In addition, the table provides information regarding key chemicals and media contributing to the hazard index and cancer risk values.

As shown in Table 5-3, total RME hazard indices for the combined residential child/adult and neighborhood recreational scenarios exceeded 1 in the Lower Basin, Kingston, the Side Gulches, Wallace, Mullan and Burke/Nine Mile. Risk drivers were arsenic and iron in yard soil and in various media (e.g., soil, sediments, and waste piles) in neighborhood recreational areas. In addition, arsenic in tap water contributed significantly to the RME hazard index for the Side Gulches.

The total hazard indices suggest that child/adult residents in these 6 areas who are also exposed to recreational media in the neighborhood might have an unacceptable threat of noncancer health effects assuming that effects from the key chemicals (i.e., arsenic and iron) are additive. However, there is no evidence that noncancer effects from exposure to arsenic plus iron are additive. Therefore, chemical-specific hazard indices were estimated for the combined residential child/adult and neighborhood recreational scenarios. The only chemical-specific hazard indices that exceeded 1 were for arsenic (added across pathways) in the Side Gulches and Burke/Nine Mile areas. Therefore, the results of the noncancer evaluation suggest that the following exposure scenarios could pose an unacceptable threat of noncancer effects for the combined child/adult residential and neighborhood recreational:

- ! Arsenic (primarily in yard soil, tap water, and soil/sediments) in the Side Gulches, and
- ! Arsenic (primarily in yard soil and waste piles) in Burke/Nine Mile.

As shown in Table 5-3, the RME cancer risks due to arsenic for the combined residential child/adult and neighborhood recreational scenarios were all in the range of 10⁻⁶ to 10⁻⁴. Lower Basin, Side Gulches and Burke/Nine Mile had the highest total cancer risks of 2 x 10⁻⁴, 3 x 10⁻⁴, and 2 x 10⁻⁴, respectively. Yard surface soil contributed most of the total RME cancer risk (Figure 5-14). Tap water also contributed significantly to the total RME cancer risk in the Side Gulches (Figure 5-14), and various recreational media (soil, sediments, and waste piles) also contributed significantly to the total RME cancer risks in Kingston, Silverton, and Burke/Nine Mile.

Other Combinations of Exposure Scenarios

For several exposure areas, the total RME hazard index and cancer risk for the child/adult residential scenario were slightly less than or equal to 1 and 10⁻⁴, respectively. For some of these exposure areas, adding neighborhood recreational exposure to the child/adult residential scenario

increased the total RME hazard index and cancer risk to levels greater than 1 and 10⁻⁴, respectively. The addition of other types of exposure to the child/adult residential scenario (e.g., ingestion of homegrown vegetables or fish, or visiting public recreational areas) might also in some cases increase the RME hazard index and cancer risk to unacceptable levels. Noncancer hazards from sport fishing were slightly less than 1 (0.9); however, the noncancer hazard from eating cadmium in vegetables was 2, and cancer risk from arsenic in vegetables was 8 x 10⁻⁵. Cadmium in vegetables is further discussed in Section 7.

5.3.4 Subsistence Risks and Hazards

This section discusses total hazard indices and cancer risk for both current and traditional subsistence exposure scenarios. For both scenarios, RME noncancer hazards were evaluated for three age groups (child of age 0 to 6 years; child/adult of age 0 to 70, and adult), and RME cancer risks were evaluated for the adult/child age group for arsenic. Tables 5-4 and 5-5 summarize the hazards and risks for the two subsistence scenarios and Figures 5-21 through 5-23 display the risks and hazards by medium for all chemicals. (See also Appendix A, Table 7 through 10 series.)

Current Subsistence Exposure Scenario

As shown in Table 5-4, total RME hazard indices were greater than 1 for each age group in the current subsistence exposure scenario, the child age group having the greatest total hazard index of 10, followed by child/adult with 4, and then adult with 3. This suggests that upon exposure to metals through all pathways there is potential unacceptable health hazard to all age groups for noncancer health effects, with children having the greatest risk.

- ! For the child age group, the key pathways contributing to total RME hazards are ingestion of arsenic and iron from surface soil, sediment, and arsenic in undisturbed surface water (Figure 5-21).
- ! For the adult/child age group, when evaluating hazards across the individual media, none of the hazard indices exceeded 1. When evaluating total hazards by
 - chemical (i.e., adding the hazards from each pathway for a particular chemical), only the total hazard index for arsenic exceeded 1 (Table 5-4).
- Ingestion of fish was the only pathway evaluated for the adult only age group. The total hazard index for fish ingestion exceeded 1, with mercury in northern pike being the greatest risk driver (Table 5-4). As was previously discussed in Section 2.2.1, whole fish tissue data is not available for use in this human health risk assessment for the tribal scenarios. Whole body metal concentrations are usually higher than fillet concentrations; thus, use of fillet data for populations which consume whole fish (tribal subsistence scenarios) likely underestimates the chemical dose from fish.

Also shown in Table 5-4 are the RME cancer risks for arsenic for the current subsistence exposure scenario. RME cancer risks 10⁻⁶ in all exposure pathways with cancer risks ranging from

approximately 1×10^{-5} to 2×10^{-4} . Total RME cancer risk is approximately 8×10^{-4} . Exposure to arsenic in sediments through dermal absorption plus ingestion contributed most of the total RME cancer risk (Figure 5-23). Ingestion of undisturbed surface water also contributed significantly to total risk.

Traditional Subsistence Exposure Scenario

As shown in Table 5-5, total RME hazard indices were greater than 1 for each age group in the traditional subsistence exposure scenario, the child having the greatest hazard of index 49. The total noncancer hazards for the adult/child and adult age groups were 10 and 21, respectively.

- ! For children, exposure to metals through all exposure pathways, except the ingestion of disturbed surface water, represents potential unacceptable risk for noncancer health effects (Figure 5-21). Ingestion of surface soil and ingestion of sediment contribute most to the total RME hazard index for the traditional subsistence exposure scenario with hazard indices of 21 and 13, respectively.
- ! For the combined adult/child age group, the total hazard index exceeded 1 for each exposure pathway except dermal absorption from surface soil and ingestion of disturbed surface water. Ingestion of surface soil is the greatest risk driver for this age group, and ingestion of water potatoes, sediment and undisturbed surface water each contribute hazard indices of 4 to the total RME hazard index (Figure 5-22). The key metals contributing to the total RME hazard are arsenic, cadmium and iron.
- Ingestion of fish was the only pathway evaluated for the adult age group. The total hazard index for fish ingestion exceeded 1, with mercury in northern pike being the most significant risk driver. As was previously discussed in Section 2.2.1, whole fish tissue data is not available for use in this human health risk assessment for the tribal scenarios. Whole body metal concentrations are usually higher than fillet concentrations; thus, use of fillet data for populations which consume whole fish (tribal subsistence scenarios) likely underestimates the chemical dose from fish.

Also shown in Table 5-5 are the RME cancer risk for arsenic for the traditional subsistence exposure scenario for the combined adult/child age group. RME cancer risks exceeded 10^{-6} in all exposure pathways, with cancer risks ranging from approximately 4×10^{-5} to 1×10^{-3} (Figure 5-23). Total RME cancer risk is approximately 4×10^{-3} , suggesting potentially unacceptable cancer risks from exposure to arsenic through all media and pathways.

The hazards from eating fish are underestimated for subsistence residents because hazard estimates are based on concentrations in fish fillets. The subsistence tribal members eat the whole fish, not just the fillets, and concentrations of metals in whole fish are greater than those in fillets. In addition, fish fillet data are from the lateral lakes, not Coeur d'Alene Lake. Sufficient fish tissue data were not available from Coeur d'Alene Lake to characterize health risks; however, tribal populations do eat fish from the lake. Therefore, tribal health hazards from eating fish from Coeur d'Alene Lake are unknown.

5.4 SUMMARY OF RISK CHARACTERIZATION

The results of the risk characterization indicate that some exposure areas could pose an unacceptable threat of noncancer effects for some individuals and exposure media under the RME condition. These include (1) young children exposed to arsenic in yard soil in the Lower Basin, the Side Gulches, Osburn, Mullan, and Burke/Nine Mile, (2) young children exposed to iron in yard soil in the Lower Basin, (3) children/adults exposed to arsenic in yard soil and tap water in the Side Gulches, (4) young children and children/adults ingesting cadmium and zinc in groundwater in Burke/Nine Mile in the future (groundwater at Burke/Nine Mile is not currently used as a drinking water source), (5) young children and children/adults ingesting cadmium in homegrown vegetables, and (6) all residents and pathways for subsistence lifestyles.

Cancer risk estimates exceeded 1 x 10^{-6} for all individuals in all exposure areas under the RME condition. Most areas also had cancer risk estimates exceeding 1 x 10^{-6} for all individuals under the CT condition. Only one scenario (RME condition for residents in the Side Gulches) had a cancer risk exceeding 1 x 10^{-4} . For the four residential areas with the highest cancer risks (Lower Basin, the Side Gulches, Osburn, and Burke/Nine Mile), the incremental increase in risk over that due to background concentrations is approximately 7 x 10^{-5} for soil exposures.

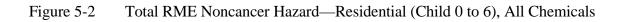
Arsenic was the only carcinogenic COPC evaluated at the site. For residential scenarios, yard surface soil contributed the most to cancer risk. For residents in the Side Gulches, tap water also contributed significantly to cancer risk. Although tap water was not the primary contributor to cancer risk for residential scenarios, RME cancer risk estimates for tap water exceeded 1 x 10⁻⁶ in all exposure areas. The risk is almost entirely due to selected high concentrations of arsenic in scattered private wells. For the Burke/Nine Mile future residential scenario, groundwater contributed nearly all of the cancer risk.

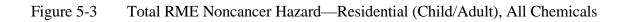
Depending on the exposure area, one or more of various media (upland surface soil, soil/sediments, sediments, or waste piles) contributed the most to cancer risk for recreational visitors. Although surface water was never the primary contributor to cancer risk, RME cancer risk estimates for "disturbed" surface water exceeded 1 x 10⁻⁶ for recreational scenarios in several exposure areas. Surface/subsurface soil contributed all of the cancer risk for construction workers.

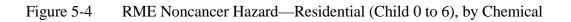
Surface soil and sediment contributed the most to hazards and cancer risks for the subsistence scenarios. The current subsistence scenario had similar hazards to those found for the highest residential child exposures. Cancer risks were higher for the current subsistence scenario, but close to those for the highest residential exposures. Hazards and risks for the traditional subsistence scenario were higher than those for the residential scenario by an order of magnitude. For the current subsistence scenario, arsenic and iron were the only chemicals with hazard quotients greater than 1 (also similar to residential hazards). For the traditional scenario, mercury in fish, manganese in soil and sediment, and cadmium in water potatoes also had hazard quotients greater than 1 in addition to arsenic and iron. Hazards from mercury in fish are likely underestimated for subsistence tribal members because they eat the whole fish, not just fillets.

Combinations of the exposure scenarios described above (e.g., child/adult residential plus neighborhood recreational) would result in hazard/risk estimates that are higher than those discussed in this summary. However, combining the risk and hazard numerical results from the scenarios probably overestimates the total numerical hazard/risk for actual residents. For example, child/adult residents are assumed to spend 24 hours/day, 350 days/year at the residence. Assuming that they also regularly spend several hours/day at a neighborhood or public recreational area or are occupationally exposed results in "double counting" (exposure for more than 24 hours/day), which will overestimate hazard/risk. However, it is clear that many of these additional exposure pathways could result in higher total risks than those shown for residential individuals.

Figure 5-1 Summary of Total RME Noncancer Hazard







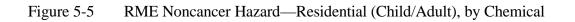


Figure 5-6 RME Noncancer Hazard—Future Residential, Burke/Nine Mile

Figure 5-7 Total RME Noncancer Hazard—Neighborhood Recreational (Child 4 to 11), All Chemicals

Figure 5-8 RME Noncancer Hazard—Neighborhood Recreational (Child 4 to 11), by Chemical





Figure 5-11 RME Noncancer Hazard—Occupational (Adult), by Chemical

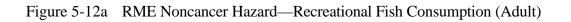


Figure 5-12b RME Noncancer Hazard—Residential Vegetable Consumption

Figure 5-13 Summary of RME Cancer Risk

Figure 5-14 RME Cancer Risk—Residential

Figure 5-15 RME Cancer Risk—Future Residential, Burke/Nine Mile

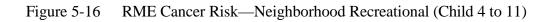


Figure 5-17 RME Cancer Risk—Public Recreational

Figure 5-18 RME Cancer Risk—Occupational (Adult)

Figure 5-19 RME Noncancer Hazard—Residential Plus Neighborhood Recreational, All Chemicals

Figure 5-20 RME Cancer Risk—Residential Plus Neighborhood Recreational

Figure 5-21 Total RME Noncancer Hazard—Current and Traditional Subsistence Exposure Scenarios, All Chemicals (Child Age 0 to 6 years)

Figure 5-22 Total RME Noncancer Hazard—Current and Traditional Subsistence Exposure Scenarios, All Chemicals (Adult/Child)

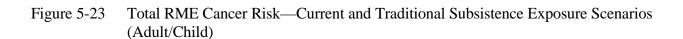


Table 5-1 Summary of Hazard/Risk Estimates and Risk Drivers

Table 5-1 continued

Table 5-1 continued

Table 5-1 continued

Table 5-1 continued

Table 5-2 Chemicals With Hazard Indices Greater Than 1

| Exposure Scenario/Receptors | Chemical | Hazard Index | | | | |
|--------------------------------------|---------------|--------------|--|--|--|--|
| Lower Basin | | | | | | |
| RME residential child | Arsenic, iron | 2, 2 | | | | |
| Side Gulches | | | | | | |
| RME residential child | Arsenic | 4 | | | | |
| RME residential child/adult | Arsenic | 2 | | | | |
| Osburn | | | | | | |
| RME residential child | Arsenic | 2 | | | | |
| Burke/Nine Mile | | | | | | |
| RME current/future residential child | Arsenic | 2 | | | | |
| RME future residential child | Cadmium, zinc | 17, 4 | | | | |
| RME future residential child/adult | Cadmium, zinc | 9, 2 | | | | |
| Mullan | | | | | | |
| RME residential child | Arsenic | 2 | | | | |
| Homegrown Vegetables | | | | | | |
| RME residential child | Cadmium | 2 | | | | |
| RME residential child/adult | Cadmium | 2 | | | | |

individual pathway not combined with hazards from other exposure scenarios or receptors. See text discussion.

Table 5-3 Summary of Hazard/Risk Estimates for Combined Child/Adult Residential and Neighborhood Recreational Scenarios

| Total Hazard/Risk | | RME Risk Drivers | | | | |
|-------------------|----------|---------------------------|---------------------------|---------------------------|--|--|
| RME | | Arsenic | | Iron | | |
| | | НІ | CR | HI | | |
| HI | CR | (Media) | (Media) | (Media) | | |
| Lower Basin | | | | | | |
| 2 | 2E-04 | 1 | 1E-04 | 0.7 | | |
| | | (Yard soil/soil/sediment) | (Yard soil) | (Yard soil/soil/sediment) | | |
| Kingston | | | | | | |
| 2 | 1E-04 | 1 | 8E-05 | 1 | | |
| | | (Yard soil/soil/sediment) | (Yard soil/soil/sediment) | (Yard soil/soil/sediment) | | |
| Side Gulo | | | | | | |
| 4 | 3E-04 | 2 | 3E-04 | 0.7 | | |
| | | (Yard soil/tap | (Yard soil/tap water) | (Yard soil/soil) | | |
| | | water/soil/sediment) | | | | |
| Osburn | | | | | | |
| 1 | 1E-04 | _ | 1E-04 | _ | | |
| | | | (Yard soil) | | | |
| Silverton | | | | | | |
| 1 | 2E-05 | _ | 2E-05 | _ | | |
| | | | (Yard soil/upland surface | | | |
| | | | soil) | | | |
| Wallace | | | | | | |
| 1 | 8E-05 | _ | 6E-05 | _ | | |
| | | | (Yard soil) | | | |
| Mullan | | | | | | |
| 1 | 1E-04 | _ | 1E-04 | _ | | |
| | | | (Yard soil) | | | |
| Burke/Nine Mile | | | | | | |
| 3 | 2.00E-04 | 2 | 2E-04 | 0.4 | | |
| | | (Yard soil/waste piles) | (Yard soil/waste piles) | (Yard soil) | | |

Notes:

Bold value indicates HI exceeds 1 or CR exceeds 1E-06.

— - not a risk driver

CR - cancer risk

HI - hazard index

RME - reasonable maximum exposure

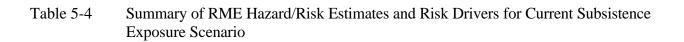


Table 5-5 Summary of RME Hazard/Risk Estimates and Risk Drivers for Traditional Subsistence Exposure Scenario